

AMENDMENTS TO THE SPECIFICATION

Please amend the paragraph beginning on page 7, line 3 as follows:

with the proviso that said peptide is not the peptide having the following sequence:

IETWILRHP (SEQ ID NO:29).

Please amend the paragraph beginning on page 7, line 5 as follows:

According to another advantageous embodiment of the invention, said peptide has the following sequence: IETWILRHP (SEQ ID NO:29).

Please amend the paragraph beginning on page 17, line 29 as follows:

- Figure 5 shows that the nine carboxy-terminal amino acids of the M ectodomain constitute a proapoptotic sequence. (A) Amino acid sequence alignments for mutant proteins (SEQ ID NOs:31-40, in descending order), the names of which are shown on the right. (B) and (C) Transfected HeLa cells were assayed for apoptotic nuclear fragmentation after 25 hours of transfection (B) or for the early stage of apoptosis after 20 hours (C). (B) HeLa cells were stained with Hoescht 33258 and examined for chromatin condensation. C⁹⁵⁻¹¹⁴-tagged EGFP (Control; open box) served as a negative control. The percentages of fusion protein-expressing cells with apoptotic nuclei are indicated. Each experimental point represents the mean \pm the SD of results obtained from three separate chambers. Statistical analysis for fusion proteins were carried out by comparison with the control. (C). The rate of early apoptosis was analyzed by Annexin V binding, as assessed by flow cytometry analysis. Apoptosis in fusion protein-expressing HeLa cells was defined as EGFP-positive cells that bound Annexin V-APC but excluded PI. For each sample, data from 10,000 EGFP-positive cells were collected. The percentages of M¹⁻⁴⁰- and M³²⁻⁴⁰-expressing cells labeled with Annexin V are indicated (square).

Please amend the paragraph beginning on page 18, line 13 as follows:

- Figure 6 shows that the residues M-34 to M-39 contribute to the death-promoting activity of the M ectodomain. (A) Amino acid sequence alignments of M^{1-40/DEN-2} (SEQ ID NO: 31), M^{1-40/YF.17D} (SEQ ID NO: 37) and mutants M^{1-40/DEN-2} (F³⁶) and M^{1-40/YF.17D} (T³⁴, I³⁶, L³⁷, H³⁹). Identical amino acids are indicated (asterisks). The amino acid substitutions are underlined and indicated in bold. (B) After 25 hours of transfection, fusion protein-expressing HeLa cells were stained with Hoechst 33258 and examined for chromatin condensation. The percentages of fusion protein-expressing cells with apoptotic nuclei are indicated. Each experimental point represents the mean \pm the SD of results obtained from three separate chambers. Fusion proteins were compared statistically with C⁹⁵⁻¹¹⁴-tagged EGFP (Control; open box).

Please amend the paragraph beginning on page 19, line 22 as follows:

- Figure 10 illustrates the alignment of the 40 C-terminal amino acids of M protein (M ectodomain; SEQ ID NO:38) from 4 serotypes of the dengue virus (DEN-1 to DEN-4), attenuated virus YFV 17D, West-Nile virus (WNV) and Japanese encephalitis virus (JEV), and also specifically the alignment of the nine amino acids of the M ectodomain (SEQ ID NOs:39 and 40) from the same flavivirus which confer apoptotic activity.

Please amend the paragraph beginning on page 26, line 28 as follows:

The plasmids pCD72¹⁻¹¹⁸-EGFP-M^{1-40/DEN-1} were generated by amplifying the cDNA encoding the amino-terminal region of CD72 by PCR, using pCR-CD72¹⁻¹³⁶ as a template and the following primers: 5'-GAGGCGGCTAGCGCTATGGCTGACGCTATCACG-3' (SEQ ID NO:30) corresponding to the 5' end of the CD72 gene and extended by 11 nucleotides to include a *NheI* restriction site and 5'-AGACACCCGGGGATAGAGAACTCCCAGGC-3' (SEQ ID NO:24)

corresponding to nt 387-402 at the 3' end of the CD72 gene and extended by 14 nucleotides to include a *Sma*I restriction site. The PCR product was digested with *Nhe*I and *Sma*I and inserted between the *Nhe*I and *Sma*I sites of pC⁹⁵⁻¹¹⁴-EGFP-M^{1-40/DEN-1} to generate pCD72¹⁻¹¹⁸-EGFP-M^{1-40/DEN-1}.

Page 46 (Abstract), after the last line, beginning on a new page, please insert the attached Sequence Listing.